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Executive function and sleep problems in childhood epilepsy

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ABSTRACT

Pediatric epilepsy has been reported to be associated with both sleep problems and cognitive deficits. In turn, in healthy children, poorer sleep has been associated with deficits in cognitive functioning. We hypothesized that poor sleep in childhood epilepsy may contribute to cognitive deficits. Using actigraphy, we objectively measured the sleep of children with epilepsy alongside that of healthy controls. In contrast to previous reports, we did not find any differences in objectively measured sleep between children with epilepsy and healthy controls. However, significant deficits in cognitive functioning were demonstrated that were not explained by differences in sleep.

1. Introduction

The relationship between sleep and epilepsy is complicated and reciprocal [1]. Sleep promotes seizure activity in specific seizure types such as frontal lobe and rolandic epilepsy. Not all stages of sleep are proconvulsant, with epileptiform discharges occurring most commonly in non-REM sleep when the EEG demonstrates synchronized background activity [2]. Furthermore, the drowsy wake state induced by sleep deprivation increases interictal epileptiform activity [3], a fact exploited in diagnostic EEG studies. While the proconvulsant nature of non-REM sleep is the main source of sleep-related seizures, obstructive sleep apnea and its associated intermittent hypoxia may also be a factor in some patients. In adult epilepsy, obstructive sleep apnea is a common [4] and treatable risk factor for seizures [5]. The literature is contradictory in childhood. Some authors have suggested prevalence rates of sleep-disordered breathing (SDB) in childhood epilepsy of almost 40% by parental report [6], although questionnaires are notoriously unreliable screening tools in this condition [7]. Importantly, treatment of sleep disorders including SDB in childhood epilepsy may reduce seizure frequency [4,8].

Sleep has developmental importance for cortical plasticity and memory consolidation [9]. Experimental sleep restriction in school children has a direct effect on teacher-rated academic performance and children's attention [10]. We have previously demonstrated that objectively measured total sleep time predicts measures of executive function in typically developing school-aged children [11]. These findings are extended by a recent meta-analysis, which indicated that shorter sleep is associated with poorer cognitive functioning and school performance [12].

A number of studies have sought to characterize sleep in children with epilepsy and have demonstrated increased problems in a variety of sleep domains such as bedtime resistance and parasomnias [13–15], although the majority rely on subjective parental report of child sleep. We have previously reported that parental report of child sleep does not always correspond to actigraphy data. Quantitative aspects of child sleep may be better measured using actigraphy, whereas qualitative aspects are better captured via questionnaires [13]. As such, some caution needs to be exercised in interpreting these data, as previous studies in clinical samples have found inconsistencies when parental report of sleep is compared with objective methods, for example, in children with ADHD [14,15]. A few studies using polysomnography, the gold standard to objectively measure sleep, have demonstrated significant differences in the sleep architecture of children with epilepsy compared with controls, including less sleep time and a reduction in sleep efficiency [16–18]. However, polysomnographic studies are typically undertaken over a single night in a laboratory setting, compromising the ecological validity of the data that are gathered. An alternative method to objectively measure sleep is actigraphy. An actigraph is a

Abbreviations: AWMA, Automated Working Memory Assessment; CSHQ, Child Sleep Habits Questionnaire; EF, Executive function; NEPSYA, Developmental Neuropsychological Assessment 1; SDB, Sleep-disordered breathing; TEA-Ch, Test of Everyday Attention for Children.

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small, watch-like device that quantifies body movements during wake and sleep and is considered a reliable method of sleep estimation. Validation studies using polysomnography have demonstrated high sensitivity (>90%) of actigraphy to detect sleep in healthy adults [19]. A number of studies have used this methodology to measure child sleep [20–22].

Hence, while there is subjective evidence of sleep disturbance in childhood epilepsy from parental report, to date, objective evidence gathered in the child's natural sleep environment is lacking. The fact that children are reported to have both behavioral insomnias and other disparate sleep disorders, such as parasomnias or SDB, suggests that causation may be multifactorial. Psychosocial factors such as parental anxiety may play a part [23].

Children with epilepsy are at risk of a variety of neurocognitive deficits, particularly those thought to represent 'executive functions'. Significant deficits across a broad range of functions have been found in children with epilepsy aged 6–12 years and were contributing factors to poorer school performance [24].

A recent meta-analysis [12] examined the differing effect of sleep time and sleep efficiency on various aspects of cognitive functioning in children without epilepsy. Sleep time was positively associated with cognitive performance and, more specifically, with executive functioning but did not show a significant relationship with sustained attention and memory. Sleep efficiency was not significantly associated with any cognitive domains. The authors suggest that developmental neurobiology (e.g., brain immaturity) may be responsible for their findings, some of which differ from those typically found in the adult literature.

In summary, sleep problems are commonly reported in childhood epilepsy, are often overlooked in clinical practice [25], but may have a profound effect on children's daytime functioning and their epilepsy control. Despite a growing body of research using actigraphy with child populations and the advantage it offers of providing an objective measure of sleep in the child's usual sleep environment over a number of nights, we are not aware of any studies that have utilized this technology in pediatric epilepsy studies [26].

The aims of this study were as follows: 1) to objectively measure the sleep of children with epilepsy and compare with that of healthy school-aged children and 2) to ascertain if sleep problems were associated with impaired cognitive functioning. We hypothesized that children with epilepsy would have greater sleep problems and that this would be a risk factor for poorer cognitive functioning. Given the difficult and complex relationship between epilepsy and cognitive functioning, we restricted our sample to children with epilepsy of unknown etiology, attending mainstream schools and with no diagnosis of learning difficulties.

2. Methods

2.1. Participants

Children were eligible to take part if aged between 6 and 13 years with a diagnosis of epilepsy with no known cause, attending mainstream school, and not taking more than one antiepileptic drug. Children were identified by general pediatricians and pediatric neurologists in tertiary hospital settings. Invitations to participate were given or posted to eligible participants. Personal details were only passed to the research team if parents consented to receive further information. Sample size estimation was based on our data in healthy populations [27]. Using a global composite for neurocognitive measures and a total sleep disturbance score, where a median split categorized 50 children without epilepsy (6–12 years) into good and poor sleepers, we found that children in the poor sleep group had significantly worse global neurocognitive scores ($M = -1.58$, $SD = 4.48$) compared with children in the good sleep group ($M = +1.52$, $M = 4.14$) ($t = 2.58$, $p = .013$), effect size of .7. This existing cohort formed the control group [11]. For a similar effect size comparing this group with a group with epilepsy, we estimated a sample of 34 children. We aimed to recruit 40 children to allow for attrition and data loss due to equipment failure.

Children (cases and controls) were excluded if they had greater than mild learning difficulties or any significant visuoperceptual or motor disabilities likely to impair their physical ability to participate in neurocognitive testing. The cognitive tests were standardized for use with English-speaking children; therefore, English had to be the child's first or main language. Children were also excluded if they were being treated with more than one antiepileptic drug.

2.2. Materials

2.2.1. Actigraphy

Children wore actigraphs (Mini Motionloggers, Ambulatory Monitoring Inc.) on the nondominant wrist 24 h a day. It was requested that the device be worn for 7 days, although this was not always possible. These devices employ a piezoelectric beam sensor, which generates voltage each time the actigraph is moved. The actigraphs were initialized to record in zero-crossing mode, which records the number of times per epoch that the activity signal level crosses zero (or very near zero); hence, it is a measure of frequency of movement. The raw data were visually inspected to reject any epochs where the actigraph had been removed. A sleep diary was used to validate actigraphy sleep and wake times. Parents kept a record of the time the child went to bed, time of waking, and an estimate of the time it took the child to fall asleep. The activity data were analyzed using Action W2 software that employed an algorithm validated for use with children [28]. Actigraphy sleep measures of interest to this study were as follows:

1) Sleep time — total minutes scored as sleep during the sleep period. This measure excludes any periods of wakefulness.
2) Sleep efficiency — percentage of minutes scored as sleep during the sleep period (which is the time from sleep onset to sleep offset). This is a reflection of the proportion of time actually spent asleep during the sleep period and is considered the measure of sleep disturbance in this study.
3) Sleep period — total minutes from sleep onset to sleep offset and is a measure of time in bed from sleep onset.

2.2.2. Oximetry

To exclude the possibility that any deficits in executive function were the result of SDB-related hypoxia in children with epilepsy, overnight oxyhemoglobin saturation (SpO_2) was undertaken for one night using a Masimo Radical pulse oximeter (Masimo — Artemis, UK). Studies were performed at home in the child's familiar sleeping environment. Data analyses were performed with Download 2001 software (Stowood Scientific — Oxford, UK). Poor perfusion, low signal IQ, and movement artifact data were rejected. Any recordings comprising less than 5 h of artifact free data were rejected. This approach increased the likelihood that representative sleep cycles were sampled, in particular REM sleep episodes where obstructive events are most likely to occur, and is consistent with previously reported methods [29]. Analysis software yields a number of measures, but those of interest to this study were the following: mean SpO_2, minimum SpO_2 (SpO_2 nadir), number of desaturations > 4% per hour, and delta 12-s index (a measure of the variability in SpO_2). The latter is calculated as the absolute differences between successive 12-second intervals (sum of the absolute difference divided by the number of intervals measured).

For the purposes of this study, each child's oximetry reports were examined to determine the likelihood of SDB. Previously published data were used to obtain oximetry reference values [30]. Combining these data, we found that the thresholds for determination of SDB were as follows: two or more abnormal parameters = probable SDB diagnosis and one abnormal parameter (with the exception of abnormal nadir alone) = possible SDB diagnosis. The threshold oximetry values used to determine probable sleep-disordered breathing were the

following: baseline SpO_2 < 95%, SpO_2 nadir < 86%, delta 12-s index > 0.4, and desaturation 4% index > 4.

2.2.3. Executive function

A battery of standardized neuropsychological tests was administered after one week of sleep measurement. The tests assess executive functions (EFs) and are higher-order cognitions such as planning, working memory, attention, and verbal fluency. This battery of tests has been successfully used in a sample of over 50 typically developing children to demonstrate a global deficit in executive functioning related to less sleep time [27].

2.2.3.1. Attention. 'Score DT' taken from the Test of Everyday Attention for Children, TEA-Ch, [31] is a test of auditory sustained and divided attention.

2.2.3.2. Working memory. Two tasks were used from the Automated Working Memory Assessment (AWMA; [32]). The AWMA is a computer-based assessment that measures verbal short-term memory, visuospatial short-term memory, verbal working memory, and visuospatial working memory.

2.2.3.3. Inhibition. Opposite Worlds (a subtest from the TEA-Ch) is a measure of verbal behavioral inhibition.

2.2.3.4. Planning. Measured using the Tower task (NEPSY; a Developmental Neuropsychological Assessment; 35). The NEPSY Tower task is modeled on Shallices' [33] Tower of London task. This task not only is considered a measure of problem-solving and planning but also requires working memory and inhibitory control.

2.2.3.5. Verbal fluency. The verbal fluency test (NEPSY) requires the child to generate words in response to semantic categories (animals and food/drink) and phonemic categories (words that begin with the letter 's' and the letter 'f').

2.2.3.6. Processing speed. Processing speed was measured using the processing speed subtests (coding and symbol search) from the WISC-IV. For coding in version A (children aged 6–7 years), the child has to mark rows of shapes with different lines according to a code as quickly as possible. In version B (children aged 8–16 years), the child must transcribe a digit-symbol code as quickly as possible. Two minutes is allocated to complete the task, with the number of correctly marked items scored. Symbol search also has a time limit of 2 min. Children are presented with rows of symbols and target symbols (one for children aged 6–7 and two for children aged 8–16). The child must indicate (by marking a 'yes' or 'no' box) if the target symbol does or does not appear in each row.

2.2.4. The Children's Sleep Habits Questionnaire (CSHQ)

This parent report sleep-screening instrument is designed for school-aged children and is freely available. The questionnaire has eight subscales: Bedtime Resistance, Sleep-onset Delay, Sleep Duration, Sleep Anxiety, Night Wakings, Parasomnias, Sleep-disordered Breathing, and Daytime Sleepiness. Items are rated on a three-point scale ranging from "usually" if the sleep behavior occurred 5 to 7 times per week to "sometimes" if the sleep behavior occurred 2 to 4 times per week and "rarely" if the sleep behavior occurred 0 to 1 time per week. Some items are reversed before scoring so that higher scores are uniformly indicative of more disturbed sleep. Hence, higher scores on the CSHQ indicate greater sleep problems. A cutoff total score of 41 has been proposed by the authors of the questionnaire as best able to identify clinical sleep problems [34].

2.2.5. The Hague Seizure Severity Scale (HASS)

The Hague Seizure Severity Scale is a 13-item scale that measures parental perceptions of their child's seizures [35]. Scores range from 13 (low severity) to 56 (high severity).

2.3. Data analysis

Raw scores for TEA-Ch, NEPSY, and AWMA were regressed onto age and gender. A composite broad EF index was computed as a sum of standardized residuals of these cognitive subtests [24]. This composite EF index had good internal consistency (Cronbach's alpha = 0.78). Group comparisons of sleep and cognitive function between children with epilepsy and children without epilepsy were conducted using ANOVA.

3. Results

3.1. Sample

Sixty-five children were identified as eligible to take part. Of these, only 33 families agreed to contact for further information. Ten children did not participate either because they could not be contacted, because they decided not to take part after receiving further information, or because they were subsequently identified as ineligible to take part. The final sample consisted of 23 children with epilepsy. They wore the actigraphs for an average of 6 days. Three children wore the device for only 4 or 5 days, 5 children wore the device for 6 days, and 15 children wore the device for 7 or 8 days. The control group was a sample of healthy, typically developing children from a previous study [11]. Children were excluded if they had a history of head injury, any neurological or psychological condition, or learning difficulties.

3.2. Demographics

The children presented with a variety of epilepsies: childhood absence epilepsy [9], generalized tonic–clonic seizures [4], complex partial seizures [36], or not known [6]. All but one child was taking antiepileptic medication (n = 22), most commonly used was sodium valproate (n = 9). Because of the small numbers involved, drug type has not been used in any analyses. The HASS scores were available for 19 children. One child scored above 26 on the HASS indicating moderate severity, a further five children scored above 39 indicating very severe seizures. Six children were reported to have nocturnal seizures, and none were known to have regular uncontrolled seizures.

Mothers' educational level was coded on a 5-point scale (0 = no qualifications, 1 = GCSEs, 2 = A levels, 3 = bachelor's degree, or 4 = postgraduate degree). Mothers' educational level was similar across the two groups, $t = -.116$, $p < .05$.

The children with epilepsy were slightly older than the control children although this was not statistically significant ($t = 1.77$, $p = .08$). As shown in Table 1, there were similar proportions of boys and girls in the two groups ($t = 0.97$, $p > .05$). We did not find any associations between age and sleep time or sleep efficiency. As would be predicted,

Table 1
Description of the sample.

	Epilepsy	Control	*p*
N	23	50	
Boys	11	22	.764
Age in years (mean, SD)	9.97 (2.04)	9.29 (1.23)	.082
Age range	6.0–13.38	6.02–11.03	
Sleep time (min)	469.32 (46.41)	486.58 (51.41)	.174
Sleep period (min)	566.32 (50.31)	567.69 (38.38)	.899
Sleep efficiency%	83.82 (7.77)	86.20 (6.82)	.191
Mothers' education	1.84 (1.12)	1.81 (.97)	.908

Table 2
Parental report of sleep measured by the Children's Sleep Habits Questionnaire (CSHQ).

	Epilepsy N = 22	Control N = 50	*p*
Bedtime Resistance	7.00 (1.41)	7.00 (1.71)	1.00
Sleep-onset Delay	1.61 (.78)	1.76 (.78)	.46
Sleep Duration	3.91 (1.13)	4.63 (1.72)	.04
Sleep Anxiety	4.96 (1.75)	4.59 (1.17)	.30
Night Wakings	3.91 (1.31)	3.55 (.94)	.19
Parasomnias	10.00 (2.09)	9.00 (1.72)	.04
Sleep-disordered Breathing	3.70 (1.06)	3.59 (.96)	.68
Daytime Sleepiness	10.35 (3.3)	11.47 (2.92)	.15
Total CSHQ	43.09 (5.41)	43.47 (6.35)	.80

Table 3
Oximetry values for children with epilepsy.

	Mean	SD	Minimum	Maximum
SpO_2 nadir (>93%)	91.47	3.08	83.00	95.00
SpO_2 baseline (>97%)	97.58	.85	94.77	98.57
Desaturation > 4%	.65	.44	.00	1.61
Delta 12-s index (>0.4)	.26	.05	.18	.39

a younger age was correlated with an increased sleep period, although this failed to reach significance.

3.3. Sleep in children with epilepsy compared with controls

There were no significant differences between the two groups on any of the actigraphic sleep measures although children with epilepsy slept on average for 17 min less than children in the control group. There were, however, significant differences in the parental report of the child's sleep (Table 2). Children in the group with epilepsy had higher scores on Night Wakings, Parasomnias, and Sleep Duration subscales of the CSHQ, indicating greater problems with these three sleep variables.

3.4. Oximetry in children with epilepsy

Table 3 shows the descriptive values of the oximetry results for the children with epilepsy (n = 23). As described earlier, a probable diagnosis of SDB was determined by abnormal parameters on two or more oximetry values and a possible diagnosis of SDB on the basis of one abnormal parameter (with the exception of abnormal nadir alone). Visual inspection of the overnight traces was also undertaken by a clinician familiar with analysis of clinical studies. Applying these criteria to the subsample with oximetry data available, we categorized two children as having a possible diagnosis of SDB.

3.5. Comparison of cognitive functioning in children with and without epilepsy

As shown in Fig. 1, after controlling for age and gender, children with epilepsy performed less well compared with the control group on all cognitive tests. Attention (Score DT), spatial working memory (Spatial), and verbal behavioral inhibition (Opposite Worlds) were all significantly poorer, whereas planning (Tower) and verbal working memory (Backward Digit) just failed to reach significance. As noted above, rather than examine executive functions in isolation, we sought to explore any differences in an aggregate measure of executive function. The aggregate measure of EF was also significantly lower ($t = 2.75$, $p = .010$) in children with epilepsy (M = −2.00, SD = 4.66) compared with control children (M = 0.922, SD = 3.05).

Processing speed was also significantly lower ($t = 2.93$, $p = .004$) in children with epilepsy (M = 92.83, SD = 15.74) compared with control children (M = 103.26, SD = 13.10), see Fig. 2.

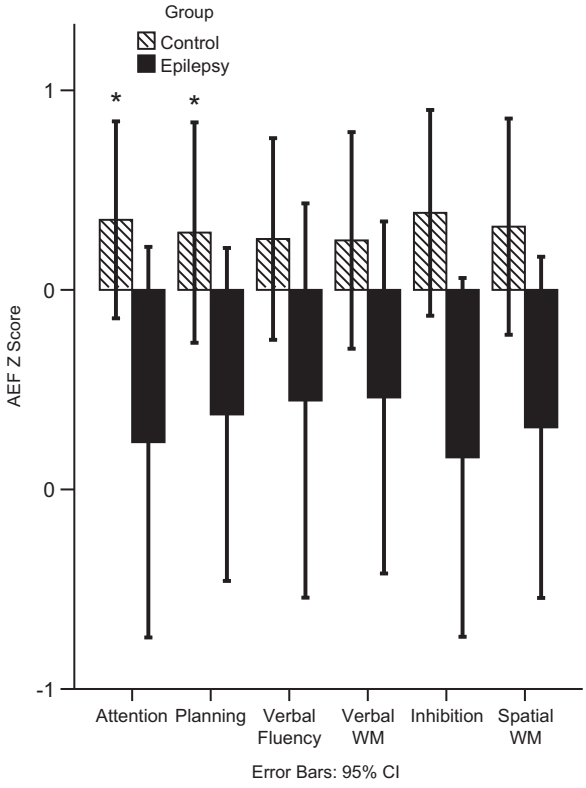

Fig. 1. Comparison of executive function (EF) domains and aggregate scores in children with epilepsy and in children without epilepsy (*$p < .05$).

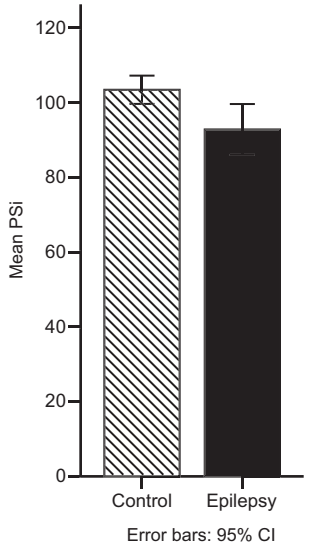

Fig. 2. Processing speed (PSi) in children with epilepsy compared with controls.

3.6. Relationship between sleep, epilepsy, and cognition

Although we did not find increased sleep problems in our sample of children with epilepsy, we still explored whether lower neuropsychological performance might be associated with increased sleep problems. Sleep time did not predict any EF measures in isolation after controlling for age, gender, and mothers' education. Further regression analyses were conducted to predict the aggregate of EF from total sleep time. As shown in Table 4, after controlling for mothers' education, less sleep time was a significant predictor of poorer EF in the control group but not in children with epilepsy.

We conducted a separate regression analysis to examine the relationship between sleep and processing speed. As shown in Table 5, less sleep time significantly predicted poorer processing speed in the control group and was in the same direction in children with epilepsy although not statistically significant.

3.7. Comparison of parent-reported sleep (CSHQ) and actigraphy data

After controlling for age, parent report of sleep duration was significantly correlated with actigraphy sleep period ($r = -.423$, $p = .050$) but not with actigraphy sleep time ($r = -.089$). Parent report of sleep latency was also significantly correlated with actigraphy sleep latency ($r = .540$, $p = .009$).

4. Discussion

The aims of this study were to characterize sleep in children with epilepsy and to assess whether sleep problems were related to poorer cognitive functioning. Based on previous research [37,38], we hypothesized that children with epilepsy would have increased sleep problems compared with healthy controls. Using a well-validated objective method of sleep measurement, we did not find any significant differences in the sleep time or sleep efficiency of children with and without well-controlled epilepsy of unknown etiology. Nonetheless, consistent with previous research, parents reported more problems with sleep in children with epilepsy. However, our data suggest that while parents are accurate at reporting time in bed, they may be less accurate at reporting actual sleep time. The mismatch between parental report and objective sleep measures is intriguing, has been noted in other settings, and could be attributed to increased parental anxiety [15]. However, while actigraphy can objectively measure simple temporal aspects of sleep, it is not designed to assess other aspects that may be reported by parents, for example, nocturnal enuresis. In balance, however, our findings indicate that children with uncomplicated and well-controlled epilepsy are not at greater risk of sleep problems, highlighting the importance of recognizing the spectrum of difficulties that present in pediatric epilepsy.

Table 4
Linear regression predicting AEF from sleep.

	B	Beta	ß	t	R2	Δ R2	F	p
Epilepsy								
Mothers' education	.41	1.00	.09	.41	.01		.11	.751
Sleep time	−.03	.02	−.32	−1.40	.11	.10	2.04	.172
Controls								
Mothers' education	.82	.30	.36	2.77	.16		8.99	.005
Sleep time	.03	.01	.40	3.11	.31	.16	9.66	.003

Table 5
Linear regression to predict processing speed from sleep.

	B	Beta	ß	T	R2	Δ R2	F	p
Epilepsy								
Mothers' education	−.56	3.14	−.04	−.18	.00	.00	.01	.919
Sleep time	.10	.07	.31	1.36	.10	.10	1.84	.193
Controls								
Mothers' education	−.23	1.31	.10	.71	.02	.00	.07	.371
Sleep time	.11	.03	.43	2.99	.19	.18	8.96	.005

As in previous research, children with epilepsy had significantly poorer cognitive functioning compared with healthy controls. In this study, we strengthened our findings by aggregating measures of EF as well as by studying individual EF dimensions. There were significant differences between the two groups for several of the individual EFs and for the overall aggregate. This is not unexpected given the previous findings; however, given that the children with epilepsy were all attending mainstream school and none had a statement of special educational needs, indicating learning difficulties, this finding is of high importance to clinicians. It suggests that these children may be at a disadvantage and that school performance may be compromised, although this may not be sufficient enough to warrant additional help from learning support systems in schools. There are several explanations for the neurocognitive deficit, and it was beyond the scope of this study to identify candidate mechanisms. However, it is known that antiepileptic medication may affect cognition; the impact of past seizures on the developing brain needs to be considered, as does the very fact of epilepsy as a marker for underlying neural damage. Furthermore, neither actigraphy nor the CSHQ provide a reliable measure of excessive daytime sleepiness (EDS), which is also known to have an impact on cognitive functioning. Future studies could usefully assess daytime sleepiness with an objective neurophysiological measure such as the mean sleep latency test.

Although we did not find differences in the sleep time or sleep efficiency between the two groups, as previous research has demonstrated an association between sleep and cognitive function in children, analyses of sleep and the aggregate EF and processing speed were conducted. Our analyses showed that in children with epilepsy, sleep did not predict either the aggregate measure of EF or processing speed. In contrast, in the control group, less sleep time was a significant risk factor for both poorer AEF and poorer processing speed. This is surprising as our clinical sample was a select group of children, purposively sampled to exclude children with learning difficulties or complex epilepsy.

4.1. Limitations of the study

It is likely that sample size was a key limitation in this study as our statistical analysis indicated that 34 children would be required to

detect a difference. Our sample size of 23 gave us only power of .28 to detect any difference. A further limitation is potential sampling bias. Only 23 of the potential 65 potential participants were studied. Sampling bias tends to skew the population towards a sample with greater difficulties in the area of study. As both sleep and neuropsychological function were highlighted in recruitment literature, it would be predicted that sampling bias would select children with difficulties in these areas. The lack of differences in sleep between the children with epilepsy and healthy controls suggests that families did not self-select on this characteristic, although bias remains a possibility. We sought to focus the study on children with no apparent learning difficulties and on anti-epileptic drug monotherapy; this resulted in a sample with a mixed profile of epilepsies. Future studies using similar methods but in a larger, more inclusive sample of children with epilepsy could more confidently resolve these uncertainties. A larger sample would also enable comparisons across different types of epilepsies, such as those with uncontrolled or nocturnal seizures. Future studies should measure such details in order to assess the potential impact these may have on sleep.

Although we were unable to demonstrate any differences in the sleep quality and quantity of children with and without epilepsy, actigraphy is unable to provide information about sleep architecture, specifically the time spent in different stages of sleep [26]. As yet, the mechanisms underlying the relationship between sleep and neurocognition are poorly understood; hence, it is possible that in children with epilepsy, there may be subtle and complex differences in their sleep architecture that may have detrimental effects on cognitive performance. Further research using polysomnography to measure sleep architecture may answer these questions.

The lack of objectively measured sleep problems in the children with well-controlled epilepsy of unknown etiology is encouraging. Further work could usefully explore the dissonance between objective measures and parental report, particularly as clinical management of sleep problems often relies on parental history alone.

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